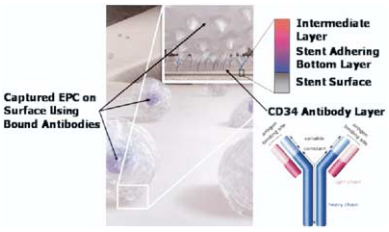
Inside This Issue of JACC

MAY 17, 2005, VOLUME 45, NO. 10



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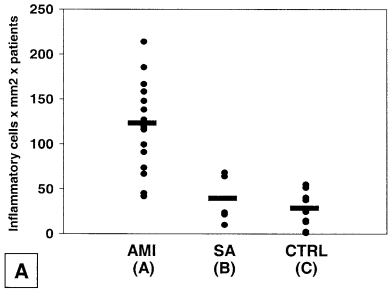
Interventional Cardiology

Novel Stent Captures Progenitor Cells to Speed Healing

The use of drug-eluting stents has reduced the neointimal hyperplasia that results in restenosis, primarily by inhibiting the body's natural healing activity through the use of cytostatic or cytotoxic compounds. On the other hand, quickly re-establishing a confluent layer of endothelial cells may suppress neointimal hyperplasia and reduce the incidence of stent thrombosis. Aoki and colleagues report a First In Man study of a stent engineered to quickly establish a healthy endothelial lining. This is achieved by capturing circulating endothelial progenitor cells through the use of antibodies attached to the surface of the stent. This pilot study demonstrates that the hypothesis seems sound. No episodes of acute thrombosis occurred and only one patient required target vessel revascularization. Future studies are ongoing using a slightly improved manufacturing method and larger patient volumes to allow meaningful comparisons of clinical utility. **See page 1574.**



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Plaque Rupture and Myocardial Infarction

Histologic Evidence of Widespread Inflammation During Myocardial Infarctions

Multiple lines of evidence suggest that acute myocardial infarctions (AMI) are associated with inflammation in the coronary vasculature. Whether this inflammation is confined to the infarct-related artery or is more widespread is not known. In this study, autopsy specimens were obtained from patients dying of either AMI or from other causes in subjects with either stable angina or no known history of coronary artery disease. The coronary arteries were dissected and serially examined for plaque morphology, thrombus formation, and evidence of active inflammation. Subjects dying from AMI were found to have on average seven additional "vulnerable" plaques; the plaques that were present also had more macrophages and lymphocytes. They conclude that in patients dying from AMI the degree of inflammatory infiltration of plaques throughout the coronary tree is three to four times greater. **See page 1585.**

Atherosclerosis

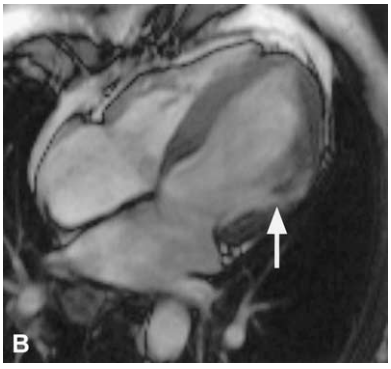
Low Testosterone Levels Correlate With Carotid Thickness

Epidemiologic evidence has consistently shown that males have higher rates of atherosclerosis than females; this led to the hypothesis that estrogen was protective and/or testosterone was atherogenic. Estrogen replacement studies have cast doubt on the protective effect of estrogen; other research has shown that high levels of testosterone may be protective, although animal model evidence is conflicting. This study examined the carotid artery intima-media thickness and levels of male sex hormones in over 200 Norweigan subjects. Andropausal men, those with low testosterone and high luteinizing hormone, had increased common carotid thickness. Intima-media thickness was found to correlate inversely with testosterone and directly with luteinizing hormone, even after adjustment for known cardiovascular risk factors. Thus, this study suggests that testosterone may be protective, possibly through its conversion to estrogen, although more research is needed. **See page 1603.**

Cardiovascular Risk

The Neutrophil to Leukocyte Ratio Predicts Future Cardiac Events

Inflammation is increasingly recognized as an important cardiac risk factor. The absolute white blood cell (WBC) count has been validated as a marker of increased risk, but whether an analysis of the cellular subpopulations would increase the accuracy is unknown. Horne and colleagues studied the total WBC count along with subpopulations in over 3,000 patients referred for coronary angiography. The total WBC count was confirmed to independently predict death/myocardial infarction, but both a high absolute neutrophil count and a low leukocyte count had better predictive power. A ratio of approximately 5:1 neutrophils to leukocytes was found to be the best predictor of future cardiac events. The pathologic mechanism is unclear, but neutrophils are known to infiltrate atherosclerotic plaques and may lead to plaque instability via the release of superoxide radicals and proteolytic enzymes. **See page 1638.**



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Cardiac Imaging

MRI Can Detect Cardiac Involvement in Sarcoidosis

The majority of deaths from sarcoidosis result from cardiac complications. The standard criteria for diagnosing cardiac involvement in a patient with sarcoidosis can either be made by endomyocardial biopsy or clinically through a combination of electrocardiogram and either echocardiographic or scintigraphic studies. Smedema and colleagues studied the accuracy of cardiac magnetic resonance imaging (MRI) to detect cardiac involvement in 58 subjects with sarcoidosis in comparison to standard techniques. All subjects with guideline-defined cardiac involvement had late gadolinium enhancement on cardiac MRI; several additional subjects were found to have small areas of enhancement that likely represented subclinical involvement. Cardiac MRI appears to be able to detect small myocardial lesions which are not detectable by other methods. **See page 1683.**